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Social networks, high-risk anal HPV, and co-infection with HIV in young sexual minority men and their network members

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Abstract

Objectives—Young sexual minority men (SMM) exhibit a high prevalence and incidence of high-risk genotypes of human papillomavirus (hrHPV) anal infections and a confluence of a

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Contributorship Statement:

K. Fujimoto initiated the project, framed the paper, designed the social network models, and co-led the writing of the article with A. Khanna. A. Khanna specified the initial social network models and analyzed the data, reported the preliminary results, and co-led the writing of the paper. A.G. Nyitray and J.A. Schneider designed data collection, framed the paper, interpreted the results, and contributed to the implications of the results. J.C. Kuo specified, conducted, and revised the social network models and reported the finalized results. A.R. Giuliano conducted HPV sequencing, genotyping, and interpreted the results. E.Y. Chiao interpreted the results and contributed to the implications and discussion. L.-Y. Hwang and J. Zhao collected, processed, and managed the specimens and data. All authors substantially contributed to the study, engaged in the writing, and approved the final version.

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Competing interests

None declared.

Ethics approval

This study was approved by UTHealth Committee for the Protection of Human Subjects: HSC-SPH-12–0830.

high prevalence of HIV and rectal sexually transmitted infections (STIs). Social determinants of health (SDOH) are linked to social network contexts that generate and maintain racial disparities in HIV and STIs. A network perspective was provided to advance our knowledge of drivers of genotype-specific hrHPV infection and co-infection with HIV. The study also examined whether socially connected men are infected with the same high-risk HPV genotypes and, if so, whether this tendency is conditioned on co-infection with HIV.

Methods—Our sample included 136 young SMM of predominantly Black race and their network members of other races and ethnicities, aged 18–29 years, who resided in Houston, TX, U.S. These participants were recruited during 2014–2016 at the baseline recruitment period by network-based peer referral, where anal exfoliated cells and named social and sexual partners were collected. Exponential random graph models were estimated to assess similarity in genotype-specific hrHPV anal infection in social connections and co-infection with HIV in consideration of the effects of similarity in sociodemographic, sexual behavioral characteristics, SDOH, and syphilis infection.

Results—Pairs of men socially connected to each other tend to be infected with the same hrHPV genotypes of HPV-16, -45, and -51, or HPV-16 and/or -18. The tendency of social connections between pairs of men who were infected with either -16 or -18 were conditioned on HIV infection.

Conclusions—Networked patterns of hrHPV infection could be amenable to network-based HPV prevention interventions that engage young SMM of predominantly racial minority groups who are out of HIV care and vulnerable to high-risk HPV acquisition.

Keywords

high-risk HPV genotype; HIV; social networks; young Black sexual minority men; social determinants of health; health disparities

INTRODUCTION

Among gay, bisexual, and other men who have sex with men (sexual minority men [SMM]), a growing number of studies on HPV molecular epidemiology have provided empirical evidence of high prevalence, high incidence, and low clearance for high-risk HPV (hrHPV) genotypes.[1, 2] Among hrHPV genotypes, HIV-16 and/or -18 infection are uniquely pathogenic for progressing to anal cancer.[3–5]

HIV infection is an independent predictor for anal hrHPV and/or HPV-16 infections, possibly due to HIV-related immunosuppression effects that increase the persistence of HPV infection and/or the reactivation of latent infections (i.e., previous anal HPV infection).[6, 7] SMM who are living with HIV have higher HPV-16 and/or -18 acquisition and lower clearance rates compared to their HIV-uninfected counterparts.[2, 8–10] HIV infection also is a strong risk factor for HPV-associated squamous cell carcinomas, caused mainly by HPV-16.[6] Notably, SMM who are living with HIV tend to develop anal cytological abnormalities more frequently and at a younger age than do their HIV-negative counterparts.[3]

Young SMM (25–34 years) and Black/African American SMM are the most affected subpopulations in terms of the highest new HIV diagnoses between 2015 and 2019 in the United States (U.S.).[11] Thus, studying a young Black SMM population is highly likely to yield meaningful outcomes related to hrHPV infection and co-infection with HIV. Only a few studies have examined HPV molecular epidemiology among a young SMM population.[12–14] Further, there is a paucity of literature on anal HPV infection as associated with anal dysplasia and cancer or that addresses racial disparities with regard to Black SMM.[15] A high prevalence and incidence of anal HPV-16 and/or -18 in young SMM [13, 14] and an association between HIV and hrHPV infection in a sample of young, predominantly Black SMM [13] aligns with a high prevalence of HIV and rectal sexually transmitted infections (STIs) as well as a lower level of viral suppression.[16, 17] Sexual behavior, however, rarely explains the racial disparities in the high prevalence of HIV and rectal STIs, as young Black SMM are less likely to engage in condomless anal intercourse [17, 18] and have lower numbers of sexual partners compared to other young SMM.[16]

Social determinants of health (SDOH) are fundamental causes that generate and maintain racial disparities in HIV and STIs.[19] SDOH determine the availability or choices of older and Black sex partners from isolated neighborhoods with a high prevalence of HIV or STIs and community viral load.[18] This would serve as a proxy for the structural disparities that are presumed to produce racial disparities in hrHPV infection, co-infection with HIV, and the lower level of viral suppression.

The analysis of sexual networks in isolation, however, have limitations when examining the structural drivers of racial disparities in disease infection, as sexual networks tend to overlap with social networks in which life experiences are shared, group norms and attitudes regarding sexual behavior are formed, and normative pressures are strongly exerted.[20, 21] Moreover, social and sexual networks often overlap in their dynamism.[22] Our study posits that social networks of young Black SMM are formed from broader networks that are not exclusive of sexual partners but, rather, are inclusive of their peers of different races and ethnicities as network members in a local community. Our broader definition of social networks helps us to contextualize the temporal and high turnover of sex partners, which could be inclusive of previous sex partnerships that are no longer active or that predate future sex partnerships that would not be observed in a cross-sectional study of sex networks.

Thus, our primary objective is to take a network approach to advance our knowledge of the drivers of genotype-specific hrHPV infection and co-infection with HIV through the lens of SDOH and social network analysis. Considering that certain high-risk HPV genotypes are more commonly propagated in the sexual networks of Black SMM [15] that are often characterized by intra-racial dense sexual networks with a high prevalence of HIV and STIs, [16, 23] our study employs exponential random graph models (ERGMs) to assess whether socially connected individuals tend to be infected with the same hrHPV genotypes. Our study also considers whether this tendency is conditioned on co-infection with HIV or on viral suppression status (with consideration of other network members' sociodemographic, behavioral, and SDOH-related characteristics) in young SMM of predominantly Black race

along with their network members of other races/ethnicities in Houston, TX. This study will refer to this somewhat heterogeneous group hereafter as “young SMM (YSMM).”

DATA AND METHODS

Study setting and data collection

This study is part of a multisite longitudinal network study, the Young Men’s Affiliation Project (YMAP), conducted in Houston, TX, and Chicago, IL. In YMAP, a total of 755 YSMM were recruited from 2014 to 2016 at the baseline period in both cities, using a respondent-driven sampling (RDS) method. The eligibility criteria for YMAP participants were aged 16–29 years, were assigned male at birth and identify as a male, reported having had oral or anal sex with another male in the past year, resided in the Houston or Chicago metropolitan area, and planned to remain in their residential area for the following year.[24]

This study used a sub-sample of 140 YSMM of predominantly non-Hispanic Black race, followed by YSMM of Hispanic ethnicity, collected from the baseline cross-sectional data at only the Houston site due to the restrictions in funding for HPV testing and genotyping analysis.[13] This sub-sample of 140 men who met the criteria of belonging to longer chains that comprised more than six persons, providing biological specimens, and participating in the computer-assisted personal interview were selected for analysis in this study. For these 140 men, HPV testing and genotyping analysis (for HPV seropositive participants only) were conducted, among whom 136 were β-globin or HPV genotype positive. All study procedures and data collection instruments received approval from the Committees for the Protection of Human Subjects at the University of Texas Health Science Center at Houston.

Measures

Network measures.—Social and sexual network data in the survey were collected by asking the participants to name up to five people with whom they shared their personal information (social partners) and with whom they had anal, oral, or vaginal sex in the past six months (sex partners). Social network data were constructed by combining the peer-referral chains (from the RDS process) with data on self-reported social or sexual partners, who also were participants in the study. A matching procedure was based on the participants and their partners’ name, age, gender, and race, was employed. Additional details of recruitment, data collection, and matching procedures are found elsewhere.[25]

Biometric measures.—HPV was tested via anal swab specimens that were self-collected exfoliated cells from the anal canal.[26] All 140 specimens were tested for HPV DNA and β-globin. A polymerase chain reaction (PCR) consensus primer system (PGMY 09/11) was used to amplify a fragment of the HPV L1 gene. DNA probes labeled with biotin were then used to identify the following 36 HPV genotypes: HPV-6, -11, -16, -18, -26, -31, -33, -34, -35, -39, -40, -42, -44, and -45; -51 through -54; -56, -58, -59, -61, and -62; -66 through -73; -81 through -84; and -89 [27]. HPV positive was defined as either -globin or HPV genotype positive. Of the sample, 136 out of 140 had adequate specimens for genotyping. HPV-16, -18, -31, -33, -35, -39, -45, -51, -52, -56, -58, -59, and -68 were included in the hrHPV

group, regardless of the presence of low-risk types. Each hrHPV type is coded as a binary variable, with 1 for a positive test result, and 0 for a negative result.

HIV seropositivity was determined by a fourth-generation Alere rapid test and confirmed by a multi-spot or viral load quantitative test. Syphilis seropositivity was defined as seropositive by a fluorescent treponemal antibody (FTA) test (Immunofluorence Assay FTA-Absorption Test System, Zeus Scientific, NJ). Unsuppressed viral load was determined by 10,000 copies/mL or above viral load and was coded as 1 for unsuppressed viral load and 0 for suppressed or HIV seronegative.

Sexual behavior.—The first measure was whether the participant had at least one receptive sex partner in the past six months (1 for yes, 0 for no), among named sex partners (described above). As this measure is restricted to a maximum of five named sex partners, we also used a second measure of the number of oral or anal sex partners in the past six months (continuous with square root transform).

Current smoking.—Current smoking was measured by frequency of tobacco use in the past three months (1 for smoked once or twice or more, 0 for never smoked).

Antiretroviral therapy (ART) adherence.—ART adherence status was measured by whether the participant currently had a prescription for HIV medications (1 for yes, 0 for no).

Sociodemographics and SDOH.—Age (continuous in years), race/ethnicity (non-Hispanic Black, Hispanic regardless of race, non-Hispanic White, non-Hispanic multi-race/other race), education (1 for some college or higher, 0 otherwise), experience in housing instability (1 for experienced homelessness in the past 12 months, 0 otherwise), current employment status (1 for unemployed, 0 for employed), currently have health insurance (1 for yes, 0 for no), and incarceration history (1 for ever jailed/detained/arrested, 0 otherwise) were measured.

Analytical methods

Population estimates of hrHPV and HIV prevalence.—RDS-adjusted prevalence estimates (with confidence intervals) of individual hrHPV types and those stratified by HIV seropositive status, using Gile's sequential sampling (RDS-SS) estimators [28], were computed. RDS-SS estimates are weighted by self-reported peer network size (i.e., degree), whereby observations with lower reported degrees are weighted higher than observations with higher reported degrees. RDS-SS estimates were computed by using the RDS program in R Statistical Software (v4.0.3).

Network visualization.—Social networks in relation to specific hrHPV genotypes, using the igraph (v1.2.6) package in R Statistical Software (v4.0.3), were visualized.

Exponential random graph models (ERGMs).—ERGMs were used to make statistical inferences about local configurations that were associated with observed social networks. As our preliminary analysis, a set of ERGMs that separately includes the effect

of assortative mixing by each sample characteristic (i.e., tendency toward social connections between YSMM with similar characteristics) on the observed network was specified. The preliminary models allowed us to select a set of assortative mixing effects that should be controlled for in the subsequent models (Models 1 and 2). In addition, the preliminary models included the pure structural effects of shared partners modeled, using geometrically weighted edgewise shared partner statistics (GWESP), of having no ties (degree 0), and of having one tie (degree 1). The GWESP statistic allows us to incorporate effects of "triad closure," that is, to estimate the probability of social connections between individuals who already are connected to at least one person in common.[29]

Then, 15 separate ERGMs (Model 1) to test the association between each genotype-specific assortative mixing effect and the observed network structure to identify specific genotypes that are significantly associated with the observed network structure were specified. Model 2 allowed the assessment of whether a tendency of assortativity by genotype-specific hrHPV infection is conditioned on HIV seropositivity. More detailed information on ERGM formulation, procedures for model selection and specification, estimation, and goodness-of-fit tests are provided in the online supplemental material. The *ergm* package (v3.11.0) in the *statnet* suite (v2019.6) in R Statistical Software (v4.0.3) was used, and missing values were excluded.

RESULTS

Descriptive statistics

Table 1 presents the descriptive statistics of our study sample ($N = 136$). The study participants were between 18 and 29 years old (*mean* = 24, $SD = 3.1$ years). A majority, 110 (80.9%), of our study participants were non-Hispanic Black, 72 (52.9%) were HIV seropositive, and 53 (39.0*%*) were seropositive for syphilis (FTA).

Prevalence of hrHPV and HIV

Table 2 presents the RDS-adjusted prevalence estimates of hrHPV type by HIV status. A total of 94 individuals were positive for at least one hrHPV type (Prev. = 0.74; 95% CI = 0.61–0.87), and 60 were positive for at least two hrHPV types (Prev. = 0.58; 95% CI = 0.43–0.72). For specific hrHPV types, HPV-59 was the most prevalent (Prev. = 0.29, 95% CI = 0.14–0.43), followed by HPV-16 (Prev. = 0.22, 95% CI = 0.09–0.34) and HPV-51 (Prev. = 0.22, 95% CI = 0.10–0.34). Further, 0.31 (95% CI = 0.17–0.45) were positive for either HPV-16 or -18.

Social network visualization

Figure 1 illustrates the observed social network separately for each hrHPV genotype as well as for both HPV-16 and -18 and either HPV-16 or -18.

Among 136 participants (nodes) who shared 125 social ties, six participants were isolated nodes and, thus, were removed for the purpose of visualization. Socially connected pairs tend to be assortative in certain genotypes such as HPV-16, -45, and -51 infections, and these tendencies are tested using ERGMs.

Exponential random graph models

For the preliminary results (the table with the results is provided in the online supplemental material), significant assortative mixing was found for HIV seropositivity ($\beta = 0.517$, $p < 0.05$) and syphilis seropositivity ($\beta = 0.559$, $p < 0.05$). Significant disassortative mixing (i.e., tendency toward social connections between individuals with dissimilar characteristics) was found for age ($\beta = -0.164$, $p < 0.01$).

For Model 1, disassortative mixing by age, assortative mixing by HIV serostatus and by syphilis serostatus, and pure structural effects to be controlled for were selected to test the effects of assortative mixing by each genotype-specific hrHPV infection. The results indicated that the significant effect of assortative mixing by HPV-16 infection on the observed network ($\beta = 1.198$, $p < 0.01$). Similar tendencies as those for HPV-16 and for HPV-45 ($\beta = 1.258$, $p < 0.01$) and −51 ($\beta = 0.928$, $p < 0.05$) were found. In addition, both HPV-16 and -18 ($\beta = 1.690$, $p < 0.01$) and either HPV-16 or -18 ($\beta = 1.027$, $p < 0.01$) had significant assortative mixing effects on the observed social network. As a sensitivity analysis for Model 1, the analysis was rerun by controlling for HIV unsuppressed viral load for individuals who are living with HIV, and the results were consistent with the model, which excludes unsuppressed viral load.

Finally, the results from Model 2 indicate tendencies toward assortative mixing by co-infection of HIV with either HPV-16 or -18 ($\beta = 1.191$, $p < 0.05$), even after controlling for the main effect of assortative mixing by HPV-16 or -18 infection and all other terms under Model 1 (except assortative mixing by HIV serostatus). More detailed analysis results for Model 2 were reported in the online supplemental material.

DISCUSSION

Our results indicate that the social networks of YSMM are characterized by social connections between network members who are infected with hrHPV genotypes of HPV-16, -45, and -51. Among men who are living with HIV, social connections tend to occur between men who are infected with either HPV-16 or HPV-18. As these men cannot choose their sex or social partners based on their HPV infection status (unlike HIV serosorting), our results may represent a higher-order social force that drives a high prevalence of hrHPV, HIV, and STIs, irrespective of individuals' sexual behavior.

Interestingly, our study suggests an independent determinant of HPV-45 and -51 anal infections in the social networks of YSMM. There results indicate that close attention is required to specific HPV genotypes that may be hyper-segregated within Black or potentially other SMM of color.[15] Our findings also indicate that the social networks of YSMM are characterized by social connections between individuals with differing ages.

There are some limitations in our study. First, our study may be limited in its understanding of the impact of social network characteristics on the potential natural history of anal hrHPV infections. Second, our results may be influenced by potential missing social ties, such as missing referral ties from the RDS sampling process and missing social or sex ties

with anonymous or unidentifiable named partners in the survey, based on which our study sub-sample for HPV genotype testing was selected.

Despite these limitations, our results have implications for potential network interventions for HPV prevention. Current individualist approaches are randomly applied and driven by individual clinic visits. Such approaches do not target HPV vaccination beyond crude individual attributes, such as age and sexual orientation. Our results indicate that leveraging existing networks, starting with network referrals from individuals who are living with HIV, virally unsuppressed, or have an hrHPV type (as opposed, for example, to random dissemination of HPV education and/or vaccination for YSMM) could yield more YSMM vulnerable to HPV.

Considering that a majority of YSMM in our sample (58%) already have been infected with multiple hrHPV types, HPV vaccination before sexual debut would be essential as a primary HPV prevention. In addition, catch-up vaccination through age 26 years (if not vaccinated) still remains imperative to HPV prevention among these men and their network members, as the exposure and clearance of individual hrHPV types do not protect against subsequent re-infection or infection with different types.

In the absence of effective screening to inform pre-cancer screening programs and an elevated rate of abnormal anal cytology among Black SMM [15], we hope that our study findings have some utility to inform an effective implementation strategy as we continue to move the incidence downward in ending the HIV epidemic and co-infection with HPV in the U.S. Such prevention strategies also may be harnessed for other cancer prevention activities, including opportunities for behavioral interventions, referrals for cancer screening, and vaccine uptake to reduce racial inequities in anal hrHVPV infection and improve population health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Key messages

- Pairs of socially connected young sexual minority men tend to be infected with the same genotypes of HPV-16, -45, -51, and HPV-16 or/and -18.
- For young sexual minority men with HPV-16 or -18, social connections tend to occur between members who are living with HIV.
- A social network-based intervention is promising to increase the uptake of catch-up HPV vaccinations in young sexual minority men.

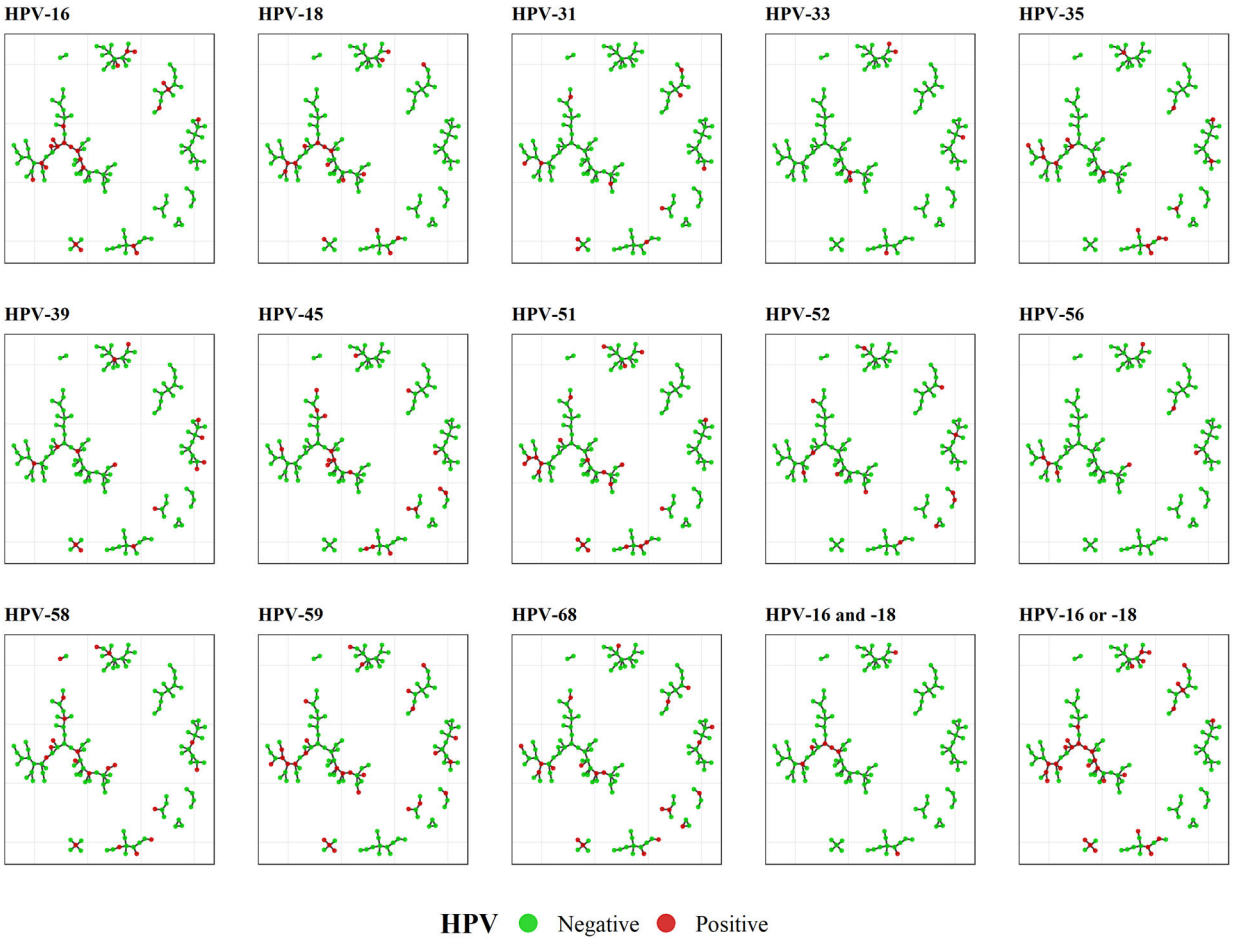

Figure 1:

Observed Social Network by Genotype-Specific High-Risk HPV Infection Status among 136 YSMM from the YMAP Cohort, 2014–2016, Houston

Note: Figure 1 presents social networks composed of peer-referral networks (via RDS sampling process) combined with social ties (participants were asked to nominate up to five people with whom they shared personal information) and sexual ties (participants were asked to nominate up to five people with whom they had anal, oral, or vaginal sex within the past six months), based on a matching procedure. Node color indicates genotype-specific high-risk HPV infection, with red as representing positive and green, negative. Socially connected pairs tend to be assortative in HPV-16, -45, and -51 infections, either HPV-16 or -18 infection, or both HPV-16 and -18 infection, and these tendencies are tested using ERGMs. Six isolated nodes were excluded.

Table 1:

Frequencies (%) or Mean (SD; Min, Max) of Characteristics of Young Black Sexual Minority Men and Network Members from the YMAP Cohort (*N* = 136), 2014–2016

Study variable	Frequency	%
HIV serostatus[a]		
Seronegative	63	46.32
Seropositive	72	52.94
Syphilis (FTA) serostatus[b]		
Seronegative	77	56.62
Seropositive	53	38.97
Unsuppressed Viral load (VL)		
HIV seronegative or HIV seropositive but with VL suppressed	104	76.47
Unsuppressed VL	32	23.53
Number of anal sex receptive partners, past 6 months (among named partners)		
None	39	28.68
At least 1 partner	97	71.32
Number of sexual partners, past 6 months (Mean = 7.16, SD = 9.23) Smoking		
Never smoked in the past 3 months	51	37.50
Smoked once or more in the past 3 months	85	62.50
Antiretroviral therapy (ART) adherence		
HIV seronegative or HIV seropositive but have ART prescription	118	86.76
HIV seropositive and do not have ART prescription	18	13.24
Age group (Mean = 24, SD = 3.1)		
18–20	16	11.76
21–25	65	47.79
26–29	55	40.44
Race/Ethnicity		
Black, non-Hispanic	110	80.88
Any race, Hispanic	17	12.50
White, non-Hispanic	4	2.94
Multi-race/other, non-Hispanic	5	3.68
Education		
High school or GED	60	44.12
Some college or more	76	55.88
Housing instability		
Never homeless in the past 12 months	105	77.21
Homeless in the past 12 months	31	22.79
Current employment status		
Currently employed (part time or full time)	98	72.06
Currently unemployed	38	27.94
Currently have health insurance		
Not currently have insurance	71	52.21

Study variable	Frequency	%
Currently have insurance	65	47.79
Incarceration history		
Never been detained, jailed, or arrested	65	47.79
Detained, jailed, or arrested at least once	71	52.21

Notes:

[a] One participant (0.7%) had an unknown value for HIV serostatus.

[b] Six participants (4.4%) had an unknown value for syphilis serostatus.

Table 2:

Frequency (*n*) and RDS-adjusted Estimate (lower, upper 95% CIs) of High-risk-HPV Genotype-Infection Statuses Stratified by HIV Serostatus of Young Black Sexual Minority Men and Network Members from the YMAP Cohort (*N* = 136), 2014–2016

	Total (*N* = 136)[a]		HIV seropositive (*n* = 72)		HIV seronegative (*n* = 63)	
High-risk HPV type	***n* Pos.**	**Prev. (95% CI)**	***n* Pos.**	**Prev. (95% CI)**	***n* Pos.**	**Prev. (95% CI)**
Any high-risk type (at least one)	94	0.74 (0.61–0.87)	56	0.93 (0.88–0.98)	37	0.55 (0.33–0.77)
Multiple high-risk types (more than one)	60	0.58 (0.43–0.72)	39	0.79 (0.67–0.91)	20	0.37 (0.16–0.58)
Both HPV-16 and -18	8	0.12 (0.01–0.23)	8	0.24 (0.04–0.45)	0	0.00 (0.00–0.00)
Either HPV-16 or -18	35	0.31 (0.17–0.45)	25	0.52 (0.32–0.72)	10	0.11 (0.01–0.22)
Any 9-valent vaccine type[b]	83	0.61 (0.46–0.75)	54	0.90 (0.83–0.97)	28	0.32 (0.13–0.51)
HPV-16	25	0.22 (0.09–0.34)	19	0.39 (0.18–0.60)	6	0.05 (0.00–0.12)
HPV-18	18	0.21 (0.08–0.35)	14	0.37 (0.16–0.59)	4	0.06 (0.00–0.14)
HPV-31	12	0.11 (0.02–0.20)	7	0.10 (0.00–0.19)	5	0.12 (0.00–0.27)
HPV-33	7	0.06 (0.00–0.12)	5	0.08 (0.00– 0.16)	2	0.04 (0.00–0.12)
HPV-35	17	0.20 (0.06–0.34)	12	0.31 (0.09–0.53)	4	0.09 (0.00– 0.24)
HPV-39	15	0.13 (0.03–0.22)	10	0.21 (0.04–0.38)	5	0.05 (0.00–0.10)
HPV-45	19	0.20 (0.07–0.32)	12	0.34 (0.13–0.55)	6	0.05 (0.00–0.13)
HPV-51	22	0.22 (0.10–0.34)	14	0.26 (0.10–0.42)	8	0.18 (0.01–0.36)
HPV-52	13	0.08 (0.00–0.17)	8	0.13 (0.00– 0.30)	5	0.04 (0.00–0.08)
HPV-56	7	0.04 (0.00–0.09)	6	0.08 (0.00-s0.17)	1	0.00 (0.00–0.01)
HPV-58	21	0.15 (0.05–0.25)	15	0.23 (0.06–0.41)	6	0.07 (0.00– 0.16)
HPV-59	28	0.29 (0.14–0.43)	20	0.38 (0.17–0.59)	8	0.20 (0.01–0.40)
HPV-68	21	0.17 (0.06–0.29)	11	0.20 (0.03–0.37)	9	0.15 (0.00– 0.30)

Notes:

[a] Total sample consists of 1 participant with missing HIV serostatus, 72 HIV seropositive participants, and 63 HIV seronegative participants.

[b] 9-valent vaccine type includes HPV-6, -11, -16, -18, -31, -33, -45, -52, and -58. *n* Pos. indicates the number of positive cases.

Table 3:

Results of Estimate (SE) of Assortative/Disassortative Mixing Effects for Each High-Risk-HPV Genotype from Exponential Random Graph Models among Young Black Sexual Minority Men and Network Members from the YMAP Cohort (N = 136), 2014–2016

Main effect	HPV		Controlled mixing terms[a]						
			HIV		Syphilis		Age	Receptive anal sex partners (n)	
	Neg	Pos	Neg	Pos	Neg	Pos	Abs diff	None	1+
HPV-16	0.089 (0.223)	1.198 ** (0.324)	0.413 (0.252)	0.337 (0.221)	0.057 (0.228)	0.450 † (0.242)	−0.165 *** (0.042)	−0.198 (0.368)	−0.396 † (0.207)
HPV-18	0.168 (0.255)	0.845 (0.517)	0.382 (0.249)	0.389 † (0.222)	0.047 (0.227)	0.442 † (0.246)	−0.164 *** (0.042)	−0.232 (0.365)	−0.376 † (0.204)
HPV-31[b]	–	–	–	–	–	–	–	–	–
HPV-33	0.762 (0.514)	1.529 (1.135)	0.374 (0.251)	0.414 † (0.219)	0.063 (0.226)	0.437 † (0.248)	−0.162 ** (0.043)	−0.239 (0.364)	−0.365 † (0.206)
HPV-35	0.416 † (0.218)	0.627 (0.510)	0.442 † (0.247)	0.335 (0.220)	0.051 (0.221)	0.448 † (0.243)	−0.169 *** (0.042)	−0.211 (0.362)	−0.402 * (0.202)
HPV-39	0.224 (0.230)	0.550 (0.963)	0.401 (0.248)	0.384 † (0.220)	0.049 (0.224)	0.446 † (0.244)	−0.163 ** (0.042)	−0.236 (0.369)	−0.365 † (0.205)
HPV-45	0.428 † (0.260)	1.258 ** (0.447)	0.378 (0.243)	0.410 † (0.213)	0.054 (0.218)	0.407 † (0.242)	−0.163 *** (0.042)	−0.221 (0.369)	−0.368 † (0.199)
HPV-51	0.066 (0.224)	0.917 * (0.416)	0.424 † (0.250)	0.366 † (0.223)	0.011 (0.227)	0.479 † (0.249)	−0.164 *** (0.042)	−0.212 (0.371)	−0.376 † (0.203)
HPV-52	0.186 (0.279)	0.134 (1.008)	0.385 (0.249)	0.408 † (0.219)	0.042 (0.222)	0.460 † (0.247)	−0.164 ** (0.042)	−0.215 (0.371)	−0.394 † (0.207)
HPV-56	0.075 (0.350)	1.302 (1.023)	0.388 (0.250)	0.390 † (0.224)	0.044 (0.226)	0.448 † (0.240)	−0.162 ** (0.042)	−0.220 (0.371)	−0.369 † (0.203)
HPV-58	0.247 (0.210)	1.218 (0.919)	0.406 † (0.242)	0.389 † (0.218)	0.050 (0.218)	0.450 † (0.241)	−0.163 *** (0.042)	−0.233 (0.365)	−0.371 † (0.199)
HPV-59	0.013 (0.210)	0.306 (0.405)	0.402 † (0.244)	0.383 † (0.217)	0.045 (0.220)	0.451 † (0.242)	−0.163 ** (0.042)	−0.221 (0.365)	−0.374 † (0.198)
HPV-68	0.076 (0.231)	0.462 (0.503)	0.400 (0.245)	0.384 † (0.219)	0.019 (0.224)	0.468 † (0.245)	−0.163 *** (0.042)	−0.218 (0.364)	−0.385 † (0.203)
HPV-16 and -18	0.007 (0.294)	1.690 ** (0.590)	0.394 (0.251)	0.356 (0.221)	0.042 (0.220)	0.466 † (0.241)	−0.164 *** (0.042)	−0.227 (0.363)	−0.363 † (0.201)
HPV-16 or -18	0.229 (0.211)	1.027 ** (0.280)	0.407 (0.250)	0.346 (0.218)	0.039 (0.222)	0.475 * (0.242)	−0.166 *** (0.042)	−0.212 (0.363)	−0.389 † (0.202)

Notes:

[a] Controlled mixing terms are incorporated in all models; only specific HPV type varies between models. Structural configurations (degree0, degree1, GWESP) are included but not shown.

[b] Model estimate for HPV-31 failed to converge.

† $p < 0.10$,

* $p < 0.05$,

** $p < 0.01$,

*** $p < 0.001$